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Occupational
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Division

*Occupational
Health Branch*

Occupational Health in Ontario

Volume 2, Number 4, October 1981

Respiratory Protection

Fibrous Glass – Is It a Health Hazard?

Criteria for Diagnosis of Asbestosis

Malignant Mesothelioma: An Asbestos-Associated Tumour

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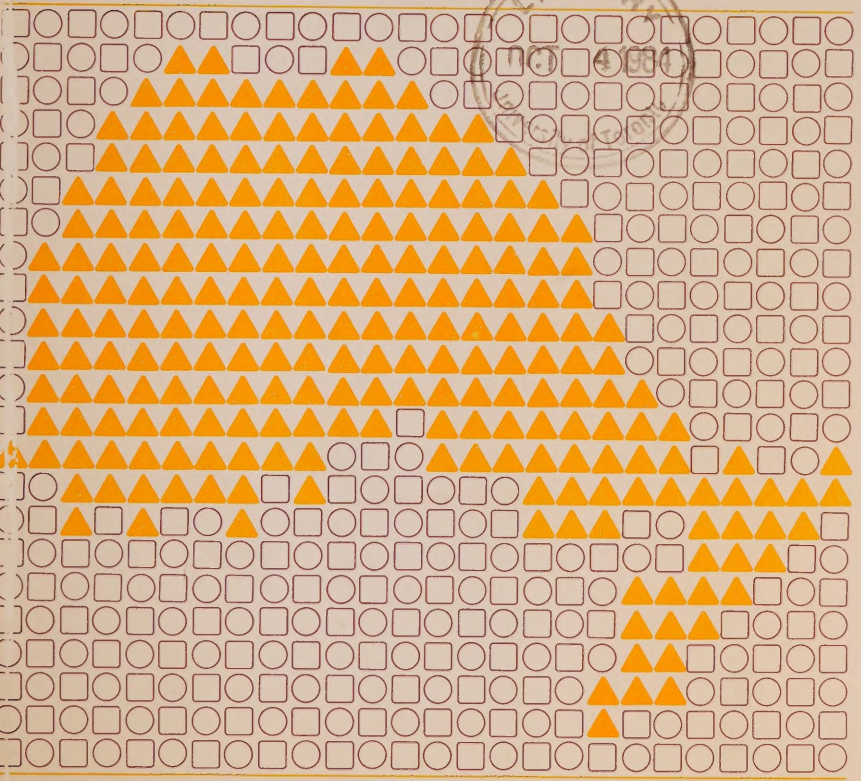

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2. Department of Health and Social Security. Notes on the diagnoses of occupational diseases, rev ed 1972; London HMSO.
3. Gamble JF, and Battigelli MC. Epidemiology. In: Clayton GD and Clayton FE. eds. Patty's industrial hygiene and toxicology. New York: McGraw Hill, 1978: Vol 1; 113-116.

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Donald A. Brown P.Eng.

Respiratory Protection*

Summary

This paper discusses the types of respiratory equipment, the protection factors and the improper use of such devices.

Respirators, as distinct from engineering or work practices, are typically only permitted under four conditions:

1) to reduce exposure during the time needed to install engineering controls,
2) to supplement engineering controls and work practices which fail to completely reduce the hazard to the permissible exposure level,
3) during activity such as maintenance and repairs when engineering controls are not a feasible means of protection, and
4) during emergencies.

Engineering controls are unquestionably the best method for effective and reliable control of hazardous substances because they act upon the source of emission. Once implemented, engineering controls protect workers permanently, if they are adequately maintained. Respirators, of course, do not eliminate the source of exposure and, by themselves, may not provide the required protection.

But because of the four situations described above, a large worker population must rely on respirators for protection.

As more stringent occupational health standards are issued, the number of workers who will rely on respirators, at least temporarily, will increase.

While the subject of respiratory protection involves many aspects, this paper will deal with three main topics:

I. Types of respiratory equipment with reference to their advantages and disadvantages. The two main categories are:
   a) air purifying
   b) atmosphere supplying
II. Protection factors.
III. Improper use (abuse).

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I. Types of Respiratory Equipment

A. Air-Purifying Respirators

These devices remove the contaminant from the breathing air before it is inhaled.

i) Particulate-Filter Respirators

These consist of a facepiece and a filter designed to give satisfactory protection against any type of particulate matter (e.g. dusts, fumes, mists, etc.).

Limitations

1. They do not provide oxygen, so they must never be used in oxygen-deficient atmospheres.
2. Particulate-filters offer no protection against atmospheres containing gases or vapours.
3. They impose some physical stress by making breathing more difficult because the filter can reduce flow of air. As a rule of thumb, when comfortable breathing is impaired because of dust build-up, the filter should be replaced.

ii) Chemical Cartridge Respirators

These are vapour and gas-removing respirators that use a cartridge attached to the facepiece containing chemicals to trap or react with specific vapours and gases to remove them from the air breathed. Some of the chemicals used in cartridges are:

i) activated charcoal (to remove organic vapours)
ii) mixture of sodium hydroxide (NaOH) and lime (CaO) (to remove acid gases)
iii) activated alumina
iv) silica gel
v) hopcalite

Two basic facepieces are available:

1) Quarter-mask – which covers the mouth, nose and the lower sealing surface between chin and mouth.
2) Half-mask – which fits over the nose and under the chin.

Limitations

1. These respirators do not supply oxygen, so they must never be worn in oxygen-deficient atmospheres.
2. Good protection may be obtained with the quarter-mask, but it is more easily dislodged than other types. Half-masks generally seal more reliably than

quarter-masks so they are preferred for use against more toxic materials.

3. Vapour and gas-removing cartridges are designed for protection against specific contaminants or classes thereof. Be sure you read the label and understand what the cartridge is designed for. A cartridge which is designed for acid gas may not give any protection against organic vapours.
4. Chemical cartridges are essentially 100 per cent efficient until the sorbents' capacity to adsorb gas and vapour or to catalyze their reaction is exhausted. Then, the contaminant will completely pass through the sorbent material and into the facepiece. This is in contrast to particulate-removing filters, which become more efficient as matter collects on them and plugs the spaces between the fibres. This difference is important to remember.
5. Water vapour reduces the effectiveness of some sorbents and increases that of others. Vapour and gas-removing cartridges must generally be protected from the atmosphere while in storage.
6. In general, chemical cartridges should not be used against gases and vapours which are:
   a) highly toxic (e.g. hydrogen cyanide)
   b) highly active with the chemical fill (nitroparaffins such as nitromethane)
   c) not removed by chemical fill (CO)
   d) severe eye irritants (SO_2)
   e) not detectable by smell or taste (e.g. bromomethane or CO)

   Odour, taste and irritation warnings are necessary to alert the wearer that the sorbent is saturated and that the contaminant is passing through the cartridge and contaminated air is being breathed.
7. They must not be used in atmospheres immediately dangerous to life or health, except for escape.

iii) Canister Respirators

These devices are also known as 'gas masks'. The basic difference between cartridges and canisters is the volume of sorbent contained, not its function. The sorbent volume of a cartridge is small, about 50–200 cm^3, so the useful lifetime is usually short, particularly in high gas or vapour concentrations. Therefore, use of respirators with cartridges is restricted to low concentrations.

Canisters have a larger sorbent volume and may be chin, front or back-mounted. Chin-style canisters have a volume of about 250–500 cm^3 and are used on full-

facepiece respirators. Front or back-mounted canisters are held in place by a harness and connected to the facepiece by a corrugated flexible breathing tube. They have a sorbent volume of 1000-2000 cm^3.

Canister-type gas masks are designed for use in atmospheres with known gas concentrations not exceeding 2 per cent by volume (20,000 ppm) or as indicated on the canister label. They are generally available for protection against organic vapours, acids, gases, or ammonia.

A very specialized type of combination particulate and vapour and gas-removing canister is the so-called 'Type N' or 'Universal' canister. It looks like a front or back-mounted canister being about the same size and held on the body in the same way. Internally, there is a great deal of difference. The distinguishing feature is that it contains several different sorbents for various vapours and gases, a catalyst, hopcalite to convert CO to CO_2, and fibrous filters for particulates.

Limitations

1. They do not provide protection against oxygen deficiency and contaminant concentrations which normally exceed 2 per cent by volume.
2. The multiple protection that the Type N canister provides has led to unfortunate, sometimes tragic, misuse. Usually, the user assumed he could get the same useful service life from a Type N canister as from an industrial canister of about the same size. Obviously this is not so.

   All Type N canisters have an indicator, usually situated behind a small window, that supposedly shows when the canister is exhausted. Actually, it indicates the condition of the drying agent upstream of the catalyst. The CO sorbent, hopcalite, is rendered useless by moisture and this indicator tells only the condition of the critical layer, not that of the acid gas, ammonia gas or organic vapour sorbent. Therefore, it cannot be used as an indicator of the overall canister condition.

   Because of the difficulties presented by the use of Type N canisters, they are being withdrawn from the market. These canisters were originally designed for emergency use, a purpose being met increasingly by atmosphere-supplying devices.
3. At very high concentrations of certain contaminants, high temperatures may develop inside the canister designed for that contaminant. When this occurs the breathing air will become hot. The wearer should leave the area at once.

iv) Powered Air-Purifying Respirators
The powered air-purifying respirator is so named because contaminated air is pulled through a filter or chemical cartridge by a small battery-operated blower and delivered to a facepiece or head covering. This type of device has a distinct advantage because it puts the facepiece or head covering under positive pressure with respect to the outside contaminated atmosphere, so that any leakage is outward from the facepiece. Thus, even if the fit is poor, contaminated air cannot enter.

Limitations
1. These respirators do not supply oxygen so they must never be worn in oxygen-deficient atmospheres.
2. The type and degree of protection depends on the air-purifying element whose protection level and useful service time depend, in turn, on its material, size and shape, and on the nature and concentration of the contaminant.
3. It is possible at very high work rates, through rapid breathing, to create a negative pressure in the facepiece, thereby potentially increasing facepiece leakage.
4. Since there is a constant air flow through the air-purifying element instead of flow only during inhalation, the useful lifetimes of sorbent canisters and cartridges attached to the blower assemblies are much shorter than when the same elements are attached to the common air-purifying respirator.

B. Atmosphere-Supplying Respirators

Supplied Air
Atmosphere-supplying respirators, instead of removing the hazardous material from the air, exclude the workplace air altogether and provide clean air from an independent source.

i) Airline Devices
They use a stationary source of compressed air delivered through a high-pressure hose.

Limitations
1. They provide a high degree of protection against gases, vapours and particulates, but they cannot be used in atmospheres immediately dangerous to life or health because the user is completely dependent on the integrity of the air supply and air source. If some-

not be able to escape from the contaminated area fast enough without endangering his life.

Note: An atmosphere immediately dangerous to life or health is an atmosphere from which a person cannot escape unprotected within a half-hour without irreversible health effects, or one that has the potential for 'obvious severe eye or respiratory irritation which would inhibit escape without injury.'

2. The trailing air-supply hose restricts the wearer's mobility.
3. In emergency situations such as fire and chemical spill, a source of compressed air is not always readily available.
4. Most plant compressed-air systems are operated with internally lubricated piston-type compressors. Oil and water mists, oil vapours, and small amounts of foreign particulate matter are present in many compressed-air systems. These contaminants should be removed by mechanical and chemical filtering. Air-line filters are available for this purpose.
5. Oil-lubricated compressors can become excessively hot, due to poor housekeeping or overdue maintenance. An oil-lubricated compressor in this condition can burn the oil, thus introducing carbon monoxide into the compressed air. Air compressors should be equipped with automatic temperature-activated shut off mechanisms or continuous carbon monoxide monitor alarm systems.

ii) Hose Masks

Hose masks supply air from an uncontaminated source through a strong, large-diameter hose to the facepiece, and do not use compressed air or any pressure regulating devices.

Those masks with hand or motor-operated air blowers have a full facepiece. The hose can be up to 300 feet long.

Hose masks without blowers must have a tight fitting full facepiece. Helmets and hoods cannot be used. The hose length is restricted to 75 feet.

Limitations

1. The hose mask with a blower cannot be used in atmospheres immediately dangerous to life or health because low air volume flow may result in a negative pressure being produced in the mask during inhalation allowing contaminated air to leak into the mask. Also, if the air hose is cut or obstructed, the user will be unprotected.
2. The trailing air supply hose may severely limit mobility.

3. A severe restriction of the hose mask without a blower is that it is limited to a maximum of 75 feet. Also, it requires the wearer to inhale against the resistance to air flow offered by the air hose, which may become significant during heavy work. Inhaling against this resistance may cause fatigue.

Self-Contained Breathing Apparatus (SCBA)

The self-contained breathing apparatus (SCBA) allows the user to carry a respirable breathing supply with him and does not need a stationary air source such as a compressor to provide breathable air.

In 1971, the National Fire Protection Agency (NFPA) recommended that fire departments no longer use the 'Type N' or 'Universal' canister type respirator because they offered no protection either in an oxygen-deficient atmosphere or against many toxic gases. As a result of this recommendation, almost all fire departments now supply their firefighters with self-contained breathing apparatus (SCBA). However, either from a lack of knowledge or a shortage of funds, most fire departments have demand-type SCBAs as opposed to the pressure-demand type units.

i) Closed Circuit SCBA

Another name for closed circuit SCBA is 'rebreathing' device. The air is rebreathed after the exhaled carbon dioxide has been removed and the oxygen content restored by a compressed oxygen source or an oxygen-generating solid. These devices are designed for one to four-hour use in toxic atmospheres.

Respiration, or breathing, involves inhalation during which air, rich in oxygen and low in carbon dioxide, is drawn into the lungs. This is followed by exhalation in which the air, containing less oxygen and more carbon dioxide owing to the gas exchange at the respiratory surface, is expelled.

Thus, in these closed rebreathing systems, it is essential that carbon dioxide be removed and oxygen added in order for the metabolic process of the body to operate.

a) Compressed Oxygen Cylinder Type

In this device, breathable air is supplied from an inflatable bag. Exhaled air from the wearer is filtered to remove carbon dioxide and the oxygen consumed is replenished from an oxygen cylinder.

The carbon dioxide absorbing reaction usually utilizes sodium hydroxide, calcium hydroxide or lithium hydroxide in granular form.

They can be used in atmospheres with oxygen deficiency and there are models available that maintain a positive pressure in the facepiece so that they can be used in atmospheres that contain toxic gases or vapours.

b) Oxygen-Generating Self-Contained Apparatus

The self-generating type uses the principle of rebreathing, but it functions in a much different manner because it has no mechanical operating components. It is commonly referred to as Chemox. This type of breathing apparatus is now manufactured for 45-minutes to four-hour durations.

This unit works on the 'lung-governing' principle in which the wearers of the apparatus, by their own breathing, regulate the oxygen supply automatically and provide the exact amount required. The oxygen-generating solid is usually potassium superoxide KO_2. The H_2O and CO_2 in the exhaled breath react with the KO_2 to release O_2.

$$2KO_2 + CO_2 + H_2O \rightarrow K_2CO_3 + 1.5O_2 + H_2O$$

This apparatus is front-mounted and consists of a hermetically sealed canister containing the chemical, a housing into which the canister is inserted, a breathing bag, breathing tubes, facepiece and harness assembly.

This self-generating type is lighter, simpler and cheaper than the tank type.

Limitations

1. Because negative pressure is created in the facepiece during inhalation (self-generating type), there is increased leakage potential. Therefore, the device should be used in atmospheres immediately hazardous to life and health only when their long-term use is necessary, as in mine rescue.
2. When using a rebreathing type, it is essential that the carbon dioxide chemical be replaced after each use. Thus, in a situation where there was long-term use and the cylinders were being replaced, it would be necessary to change the chemical also.
3. Once the canister containing the oxygen-generating chemical is opened and the reaction started, it cannot be turned off. Thus, a new canister must be installed after each use no matter how short the use was.
4. The warning device on the Chemox is normally a timer indicating the passage of time; it does not show the condition of the canister.

ii) Open Circuit SCBA

An open circuit SCBA exhausts the exhaled air to the atmosphere instead of recirculating it. A tank of compressed air carried on the back supplies air via a regulator to the facepiece. Because there is no recirculation of air, the service life of the open circuit SCBA is shorter than a closed circuit system. Two types of open circuit SCBA are available – 'demand' or 'pressure-demand'. For most fire service use, the 30-minute apparatus is used.

Testing and approval of SCBAs was originally set up by the US federal government in the Bureau of Mines, whose primary concern was miners. In 1972, The National Institute for Occupational Safety and Health took over the testing functions for all types of respiratory devices, and today approvals are issued jointly by NIOSH and Mining Enforcement and Safety Administration (MESA). However, only minor changes have been made over the years to the test requirements used by the Bureau of Mines and currently by NIOSH. Thus, the testing and approval procedures do not take into consideration the adverse conditions found by firefighters.

The user should not expect to obtain exactly 30 minutes service life from this apparatus on each use. The duration of the unit will depend on factors such as:

a) the user's degree of physical activity
b) the user's physical condition
c) the degree to which the user's breathing is increased by excitement, fear or other emotional factors
d) whether or not the cylinder is fully charged at the start
e) the degree of training or experience which the user has had with this, or similar, equipment
f) the atmospheric pressure; if used in pressurized tunnel or caisson at 2 atmospheres (15 psi gage), the duration will be one-half as long as when used at 1 atmosphere.

a) Demand SCBA

Air at approximately 2000 psi is supplied to the regulator through the main value. A bypass valve passes air to the facepiece in case of regulator failure. Downstream from the main valve, a two-stage regulator reduces the pressure to approximately 50-100 psi at the admission valve, which is actuated by movement of a diaphragm and its associated levers. The admission valve stays closed as long as positive pressure in the facepiece (during exhalation) presses the diaphragm away from the valve assembly. Inhalation creates negative pressure in the facepiece, and the diaphragm contracts, opening the ad-

other words, air flows into the facepiece only on 'demand' by the wearer, hence the name.

Limitations

1. During inhalation, there is a negative pressure in the mask, so if there is leakage in the facepiece, contaminated air can enter the mask and be breathed by the user.
2. In a demand-type SCBA, negative pressure in the facepiece during inhalation opens the demand valve. This negative pressure is approximately the same as that created in an air-purifying respirator. Therefore, leakage is inward and of the same magnitude as that in an air-purifying respirator. Recent studies show that a demand-type SCBA is no more efficient than an air-purifying respirator with the same facepiece. Therefore, a demand-type open circuit SCBA should not be used in atmospheres immediately hazardous to life or health. Like closed circuit SCBAs, they are, however, adequate against oxygen-deficient atmospheres.

b) Pressure-Demand SCBA

This unit is very similar to the demand-type except that there is usually a spring between the diaphragm and the outside case of the regulator. This spring tends to hold the admission valve slightly open, theoretically allowing continual air flow into the facepiece. This would be true except that all pressure-demand devices have a special exhalation valve that maintains about 1.5 to 3.0 H_2O positive back pressure in the facepiece and opens only when the pressure exceeds that value. This combination of modified regulator and special exhalation valve maintains positive pressure in the facepiece at all times, and the regulator still supplies additional air on 'demand'.

Because of the positive pressure, any leakage is outward so a pressure-demand SCBA is adequate for atmosphere immediately dangerous to life or health.

Limitations

1. As is the case with the demand-type, the pressure-demand respirator is bulky and heavy, and thus may not be suitable for work in confined spaces.
2. The air supply is limited to the amount in the cylinder and therefore the respirator cannot be used for extended periods without recharging or replacing the cylinders.
3. Since the facepiece is under positive pressure, any leakage increases air consumption and decreases service time.
4. One might assume that compressed oxygen could be

can not be. Minute amounts of oil or other foreign matter in the device component can cause an explosion. In general, oxygen should never be used in a device unless it is specifically designed for that purpose.

5. Never interchange demand and pressure-demand equipment. If you put a pressure-demand regulator on a demand facepiece, air will be blown right through the exhalation valve and the life of the air tank will be greatly shortened.
   If you put a demand regulator on a pressure-demand facepiece, there will be great resistance to breathing.

Safety features typically found on all approved closed and open circuit SCBAs include:

a) pressure gauges or liquid gauges visible to the wearer that indicate the quantity of gas or liquid (air or oxygen) remaining in the cylinder;
b) remaining service-life indicators or warning devices that show when only 20-25 per cent remains;
c) fittings on devices that use compressed or liquid oxygen that are incompatible with compressed or liquid air fittings.

II. Protection Factors

The overall protection afforded by a given respirator design may be defined in terms of its protection factor (PF). The PF is a measure of the degree of protection afforded by a respirator, defined as the ratio of concentration of contaminant in the ambient atmosphere to that inside the facepiece under conditions of use. Determination of the protection factor requires quantitative performance tests of the respirator worn in a test atmosphere during exercise simulating motions made by workers.

The most common method of determining protection factors is by carrying out quantitative fit testing. The wearer first puts on a respirator which has a probe, attached to a length of flexible tubing, installed in the facepiece. The wearer then enters a chamber in which there is an atmosphere of a known aerosol. The tubing from the respirator probe is attached to instrumentation that can measure the aerosol both inside and outside the facepiece. A comparison of the two concentrations thus determines the protection factor which is really the leakage rate.

The protection factors used by OSHA in the United States are based on fit tests performed at Los Alamos Scientific Laboratory and are shown in the following Table. These protection factors are a very important part of the respirator decision logic.

Respirator Protection Factors

Type of Respirator	Facepiece pressure	Protection factor
1. Air-Purifying		
a) Particulate Removing		
single-use, dust	—	5
quarter-mask, dust	—	5
half-mask, dust	—	10
half-or quarter-mask, fume	—	10
half-or quarter-mask high-efficiency	—	10
full facepiece, high-efficiency	—	50
powered, high-efficiency; all enclosures	+	1,000
powered, dust or fume; all enclosures	+	X
b) Gas and Vapour-Removing		
half-mask	—	10
full facepiece	—	50
2. Atmosphere-Supplying		
a) Supplied-Air		
demand, half-mask	—	10
demand, full facepiece	—	50
hose mask with blower, full facepiece	—	50
hose mask without blower, full facepiece	—	50
pressure-demand, half-mask	+	1,000
pressure-demand, full facepiece	+	2,000
continuous flow, half-mask	+	1,000
continuous flow, full facepiece	+	2,000
continuous flow, hood, helmet, or suit	+	2,000
b) Self-Contained Breathing Apparatus		
open-circuit, demand, full facepiece	—	50
open-circuit, pressure-demand, full facepiece	+	10,000
closed-circuit, oxygen tank-type, full facepiece	—	50
3. Combination Respirator		
a) Any Combination of Air-Purifying and Atmosphere-Supplying Respirator		Use minimum protection factor listed above for type and mode of operation
b) Any Combination of Supplied-Air Respirator and an SCBA		

To apply an assigned protection factor to a particular type of respirator, one must know both the actual concentration in the work area and the established time-weighted average concentration standard or guideline or permissible exposure limit used to assess worker exposure. Multiplying the time-weighted average concentration by the respirator protection factor gives the maximum concentration of the contaminant against which the particular type of respirator may be used. If the actual concentration is less than the calculated maximum use concentration, the respirator may be used.

While the application of protection factors seems simple enough, it is important to realize that if the time-weighted average concentration or standard changes, the maximum use concentration will be affected. This is essential when comparing or referring to information published in the US, such as NIOSH Criteria Documents, which mention the type of respirator required. Let us consider a dust which is assigned a standard of 1 mg/m^3 in the US. The prescribed respirator for concentrations of up to 5 mg/m^3 would be a single-use or quarter-mask dust respirator, each of which has a protection factor of 5. However, let us assume that in Ontario, this same dust has a TWA or standard of 0.5 mg/m^3. In this case, for exposure of up to 5 mg/m^3, a half-mask dust respirator would be required. Thus, in this situation, the respirator required in Ontario would be of a higher protection factor than that required in the US for the same dust at the same concentration.

At the present time, there is confusion about the actual purpose of quantitative fit testing carried out in companies where a respirator program is in effect. An essential ingredient of proper protection by a respirator is a proper fit. This can be achieved by carrying out either qualitative or quantitative fit testing. Qualitative tests (using irritant smoke or isoamyl acetate) are more widely used; however, the major fault is that these tests require the understanding and co-operation of the user. These two essential items are not always present.

The preferred method of testing is quantitative fit testing. The major drawback is the enormous expense.

However, when quantitative fit testing is carried out, it is essential to realize that the data is collected to determine whether or not the respirator fits properly, and not what protection factors apply.

In the US, the American National Standard Institute (ANSI) Z88.2 Committee has suggested that the protection factors determined by individuals carrying out quantitative fit testing be used for respirator selection and not just for the determination of whether or not a

respirator fits properly. Thus, if this suggestion were accepted by OSHA, higher PFs would be permitted in the US for wearers who had been fitted quantitatively rather than qualitatively.

In this proposed standard, the protection factors, determined according to the fitting method used, permits an increase from 10 for qualitative fit to a maximum of 100 for quantitative fit for half-mask facepiece particulate-filter respirators, and from 100 to a maximum of 1,000 for full facepiece respirators equipped with high efficiency filters.

Many people feel that this suggested revision is a realistic standard that permits the employer a safe incentive to use quantitative fit test systems. However, it is important to realize that this standard has not yet been adopted, and the protection factors shown in the preceding Table still apply.

III. Uses and Abuses

In some instances, respirators are the only control measures that can be used to protect workers from respiratory hazards. However, in many cases, abuses prevent the worker from receiving adequate protection. Some of these abuses are demonstrated by the following cases histories:

Case History 1: The Removal of Insulation from Steel Beams in the Construction Industry

The dust produced by this operation can be very hazardous, especially if the insulation contains asbestos. In this case, since the worksite was not permanent, local mechanical exhaust was not feasible and general ventilation was not effective. Thus, respirators were used for controlling the dust exposure.

Some of the typical abuses included the wearing of non-approved respirators by workers and the failure to issue respirators to other tradesmen working in the same area. It was also very common to find respirators lying around the jobsite gathering dust which was later inhaled by the workers when they next put on these respirators.

As mentioned, the first abuse involved the wearing of non-approved respirators. While initially established as a research and education arm of OSHA, NIOSH has been named officially as the testing, approving, and certifying agency for a variety of personal safety devices, including respirators in the US. In Ontario, we have no testing and certification for respirators, and thus we normally accept the approval of NIOSH or equivalent approval agencies.

A respirator which is not approved by a recognized approval agency is considered to be a 'non-approved' respirator. In other words, the 'non-approved' respirators do not provide the same kind of protection as the 'approved' ones. When it is not possible to provide adequate engineering controls for dust producing operations, it is imperative that any persons coming into the area wear approved respirators. The fact that these people (i.e. other tradesmen, supervisors) are not directly involved in the production of dust, or may only be in the area for a short period, does not absolve them from the need to be protected from the dust by wearing a proper respirator.

Ideally, all dust exposures should be thorougly evaluated by air sampling before respiratory protection is prescribed. In the construction industry, this is not always possible since the jobsite changes so frequently that by the time the sampling is done and results are available, the job is finished. In situations such as this, when adequate information is not readily available, it must be assumed that the greatest hazard possible is present and, consequently, the use of approved respirators should be made mandatory. Often, it has been observed that the contractor awaits a directive from the government agency before providing his employees with respiratory protection in addition to the other personal protections which are provided as a rule.

Case History 2: **Abrasive Blasting**

Abrasive blasting to clean metal parts creates a very high dust exposure. Most people are aware of the hazards of this operation and it is not uncommon to see it carried out in an exhausted booth by an operator wearing an air-supplied respirator.

However, in one company it was observed that the respirator was left inside the blasting booth after the operation where it picked up the settling dust.

The suggested solution was to not provide hooks or hanging devices in the booth and to instruct the operator to store his respirator in a clean, dust-free place.

It should also be stressed that the exhaust from the abrasive blasting booth is merely to keep the dust down for visibility purposes, and that the air-supplied respirator is the device that actually protects the workers from the dust generated.

It is not possible to provide an exhausted booth for all abrasive blasting operations, e.g. the engraving of granite cemetry stones, which contain a significant

amount of silica. Some workers feel that because they are working outside, the atmosphere will provide infinite ventilation, and that a disposable respirator is adequate to protect them from the dust produced. Although the atmosphere will dilute the dust to a safe level at a distance of 50-100 feet away from the source, the dust level at the operator's breathing zone may be far too high to be controlled by a single or reusable respirator. Only an air-supplied respirator should be considered adequate.

Case History 3: **Nuisance Dust**

A worker bagging calcium carbonate was found to be wearing a nuisance dust respirator because the ventilation system had broken down and an excessive level of dust was being created. The company stated that the worker was being adequately protected since the dust produced is classified as nuisance dust by the ACGIH and therefore, there was no reason to use an 'approved' respirator.

For exposures such as these, we do not feel that nuisance respirators are a good protective device because:

a) no approval tests have ever been performed on these respirators by any authorized agency, and
b) they are specifically for nuisance dusts, hence they are not acceptable for a dust which, by virtue of the concentration and particle size, could become toxic.

Case History 4: **Look-Alike Respirators**

A major respirator producer manufactures two types of disposable units: 'approved' and 'non-approved'. To many workers, these respirators appear to be identical and we have found workers wearing non-approved respirators in asbestos and silica exposures. The obvious solution is to eliminate the non-approved respirators. However, many companies refuse to do this. The workers must therefore be trained to look for the approval number stamped on the front of the approved respirator. The nuisance or non-approved respirator has no identification number on the front.

Case History 5: **Lack of Inspection**

Many times during our investigation, workers were found to be wearing respirators with the exhalation valves jammed or locked in the open position or with rubber facepieces that were badly cracked. In both cases, dust was allowed to leak through the respirator to be inhaled by the worker. In order to avoid these leakage problems, respirators must be inspected every day and corrective maintenance carried out or new units issued.

Case History 6: **Improper Selection**

A workman entered a sewer manhole located in a street just outside a brewery. After a few minutes, he felt tired and weak. He tried to climb out of the manhole, but collapsed. A worker in the brewery, seeing this, rushed over with a respirator. The workman's helper put on the respirator and proceeded into the manhole. Within a few minutes he, too, collapsed. When these two men were finally brought out of the manhole by firefighters wearing self-contained breathing apparatus, they were both dead.

This clearly illustrates the hazard of the improper selection of respirators. In this case, carbon dioxide from the brewery had entered the sewer and displaced air. The two men died from a deficiency of oxygen. The respirator used by the rescuer, designed to remove ammonia from air, could not be expected to correct for insufficient oxygen for breathing. In this instance, an atmosphere-supplying respirator was needed.

Case History 7: **Airline Respirator**

In a plant producing brake lining, a significant exposure to asbestos was occurring at the operation where dry mix containing asbestos is hand-scooped from a tote box into a preform press hopper. Although the hopper contained some limited ventilation, the tote box was unventilated and considerable dust was produced each time the operator transferred a portion of the mix. Although the work practices definitely affected the worker's exposure, it was found that no matter how carefully the operator worked, his exposure was always above the Ontario standard of 2 fibres/cc.

As an interim measure, until improvements in ventilation and possible process changes were being implemented, the operator was required to wear a

respirator. The company felt that a continuous air flow helmet would give adequate protection based on a protection factor of 2,000.

Since tow motor skids were used to move metal tote boxes into and out of the area, it was decided that the airline hose could not be allowed to drag along the floor. Thus, a clothes line was installed above the hoppers and the airline was coiled around this to prevent it from contacting the floor.

While this system seemed good in theory, a visit to the plant by the author revealed a number of problems that were preventing the operator from receiving the theoretical protection he should have.

The first problem was that the airline became badly tangled. This resulted in the operator's having to tug and pull on the line. Thus, the respirator was a hindrance to him and, occasionally, the operator would remove the helmet.

The second problem was that because of the tangling, and the pulling by the operator, the airline was worn in places and minor leaks resulted. This reduced the flow of air to the helmet and subsequently reduced the respiratory protection.

Thirdly, although the helmets were provided with clear visors, it was common to see the visors flipped up.

It was also noted that the operators from the other shifts stored their helmets on hooks in the preform press area. This allowed dust to settle inside the helmet and cause exposure when the operator next used it.

While continuous flow airline helmets can offer a high degree of protection, the abuses which were observed, were preventing the operators from receiving adequate protection. The airline hose was too much of an inconvenience since it had to be kept off the floor. Also, there was no maintenance or inspection of the airline or helmet, which is essential. The helmets should have been stored in a clean, dust free location.

Due to the abuses and limitations of the airline respirator in this situation, the company switched over to powered air purifying respirators.

Case History 8: **Facial Hair for Firefighters**

There is a continuing debate in Ontario as to whether fire department personnel who are required to wear self-contained breathing apparatus should be allowed to have facial hair. Management maintains that since a good face seal is not possible, then for the workers'

health and safety, they should not be allowed to have facial hair. The firefighters or union say that it is an invasion of workers' rights to be told that they cannot have facial hair.

In order to ensure that bearded firefighters will be adequately protected from toxic contaminants, it is essential that they wear only pressure-demand SCBAs. This ensures a positive pressure on the facepiece. A demand-type SCBA is not acceptable if the firefighter has facial hair. This is due to the negative pressure in the facepiece and the corresponding low protection factor of only 50 as opposed to 10,000 for pressure-demand.

It is, however, essential that the firefighters understand that although the pressure-demand will likely give adequate protection because all leaks caused by the facial hair will be outward, they will not get the same service life from their tanks. Thus, 30-minute air tanks may give only 20-25 minutes of air.

Case History 9: **Do as I Say Not as I Do**

Most workers who are required to wear respirators do not want to wear them. They feel that respirators are uncomfortable, that they may interfere with communication and are a nuisance because it is necessary to take them off to eat, drink, smoke or blow their noses. The way to get worker co-operation is to offer a thorough explanation of why it is necessary to wear a respirator. If measures are being taken to correct the work situation so that workers will, in future, be allowed to work without a respirator, they should be told what these measures are. Stress should be placed on the fact that wearing respirators now is an interim measure to protect worker health.

However, it often happens that after the operator has been persuaded to wear a respirator, he sees many people who are not wearing them come into his work area. These people could include:

i) his supervisor or foreman
ii) maintenance workers
iii) government inspectors

The operator is then inclined to ask why he has to wear a respirator if others do not. It is sometimes argued by supervisors and inspectors that as they will be in the area for only a short while (up to one hour or two) their exposures, averaged over the 8-hour shift, would not be above the TWA or standard. While true, the worker usually finds this reasoning difficult to accept, and his

negative feelings towards wearing a respirator are reinforced.

The situation then develops into one in which the worker is required 'to do as I say, not as I do.' This inconsistency has a great effect on increasing a worker's reluctance to wear a respirator.

It should be the policy of every company that any person who enters, no matter for how short a time, an area of the plant where workers are required to wear respirators, also be required to wear one.

Case History 10: **Construction Workers Cutting Cement with Power Saws**

Typically, when this type of cutting operation is carried out, it is done wet. It is not uncommon to see workers perform this operation on sidewalks and streets. However, when cutting is required on cement barriers between lanes of an expressway, it is often done dry because a more portable saw is used.

When the cutting is done dry, an excessive concentration of dust is generated at the workers' breathing zone. It is very rare to see these operators wearing respirators since they feel that the fresh air around them provides infinite dilution. This is not true, and they should wear proper respiratory protection.

Conclusion

The abuses of respiratory protection can be avoided by establishing a written training manual and by providing proper supervision of all aspects of the respirator program. Without a definite chain of supervision, there is no assurance that written standard operating procedures will be followed. Therefore, responsibility for the entire respirator program should be assigned to one person, and that person should have the full support of higher level management; without it, an effective respirator program is difficult to initiate and maintain.

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Fibrous Glass – Is It a Health Hazard?*

Summary

The manufacture and occupational health hazards associated with fibrous glass are discussed.

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For centuries people have been dependent on natural fibres to produce a wide variety of goods to serve their needs. The technical know-how of the twentieth century has brought forth a variety of non-natural fibres that can be tailored to meet specific needs. One such fibre is glass.

Prior to the early 1930s, glass fibres were largely a laboratory curiosity. Beginning about 1932, fibrous glass was manufactured on a limited commercial basis for use in air filters and insulation; in 1936, a textile form of the material was developed. Present-day fibrous glass materials are manufactured into a wide variety of products suitable for specific applications. Fibrous glass products are frequently classified into two general categories: (1) thermal and acoustic insulation (wool) and (2) textile fibres. The thermal and acoustic insulation fabricated from wool is used to insulate residential and commercial buildings, appliances, aircraft, etc. By varying the compression and density, the wool is formed into such products as ceiling boards, tiles, ducts, duct liners, pipe coverings and roofing systems.

The textile fibres are woven into cloth for draperies, electrical insulation and other industrial applications. In addition, textile fibres are processed into rovings, chopped fibres, mat and yarn, and into materials for the reinforcement of rubber plastics, paper and other products.

Morphology of Fibrous Glass

Although the manufacturing technology has improved since the early 1930s, most fibrous glass continues to be produced in a fairly conventional glass furnace, using a boro-silicate batch. Current wool products that are produced by the more modern centrifugal process have an average diameter of roughly 6-12 microns, depending on the product. Some fibrous glass materials that are produced by a flame-blowing process have an average diameter range of 1–4 microns; others range from 6–9

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microns in diameter. Those with fibres within the range of 1–4 microns have been produced since the early 1940s. These fine diameter products presently account for less than one per cent of the glass fibre market. The coarser flame-attenuated products, whose fibre diameters come within the range of 6–9 microns, account for a substantially larger proportion of the total glass fibre insulation production.

In Canada, wool products with a nominal diameter of 1–4 μ are not produced.

Textile-type products, in contrast to 'wool', are produced by an entirely different process. Continuous-filament textile fibrous glass is produced by melting glass marbles in a small electric furnace located above each forming position. A more recent method of producing textile glass consists of conveying the molten glass from a large central furnace to each forming position. Streams of molten glass flowing from bushings are mechanically pulled into continuous filaments, which are drawn over a sizing applicator. The filaments are collected into strands and the strands are wound onto rotating forming tubes. Because of this processing method, the fibre diameter is quite uniform, with little deviation from the mean diameter. The nominal textile fibre diameters, other than the B fibres, run from five to approximately 15 microns, with the majority in the larger-diameter fibres. The B fibre has a nominal diameter of 3.5 microns, and this diameter accounts for less than one per cent of the fibrous glass textiles produced.

In Canada, textile fibres with a nominal diameter of 3.5 μ are not produced and textile fibres are only manufactured by the more recent direct melt method, so no glass marbles are used.

Practically all fibrous glass products are coated with binding, sizing and lubricating coatings. During the past 30 years, there has been little change in the basic chemical composition of the ingredients of insulation binding resins. The most significant change was the progressive replacement of the bonding materials, which consisted largely of phenol formaldehyde resins and insoluble wood resin by-products, by a bonding material made up mainly of phenol formaldehyde resins and nitrogen-containing components to improve the combustion resistance of the product. Originally, water-soluble resin, after cure, remains as an insoluble and infusible resin film on the fibre surfaces. The wool binder coating is cured in an oven immediately after application and prior to any handling by manufacturing workers.

Since the 1930s, high flash point parafinic mineral oils have been used in all wool products with little change in

composition or percentage of total product weight (0.25–1.0 per cent). The oil prevents the uncured resin-coated glass from sticking to the curing flights and reduces lint generation in the cured insulation.

Textile sizes or binding agents are of the starch or cationic lubricant vegetable oil type for yarns that are woven into decorative fabrics, such as curtains and drapes. This starch-base size is removed from the woven fabric by heat treatment. Textile fibres destined for reinforcement applications are coated with a polyvinyl acetate-chrome chloride or epoxy-silane size appropriate to the reinforcement application. The fibres generally used in reinforcement applications are those with average diameters above 7.5 microns.

Occupational Health Hazards

a) Fibrous Glass and the Skin

Fibrous glass is capable of producing a mechanical, transitory skin irritation that is characterized by a maculopapular eruption. The eruption is usually noted at pressure points, such as around the waist, neck and wrists, and the transitory irritation commonly decreases within three to five days of beginning work with the material.

The irritation, the symptoms of which are customarily described as a prickling, burning or itching sensation, is caused by the direct handling of the fibrous glass, and not the airborne fibres.

The ability of fibrous glass to cause transitory mechanical irritation is related to the diameter of the fibre.[1] The larger the diameter, the more likely the material is to cause irritation. Since the advent of modern centrifugal fibrizers, irritation has been reduced by improved fibrization of the glass.

During the manufacturing process, the phenolformaldehyde binder that is applied to wool-type products is cured, prior to handling, into an insoluble, infusible film. The cured binder is not a sensitizing agent. Skin sensitization to the plastic-compatible sizes can occur in some workers in the manufacturing of textile-type fibrous glass. Sensitization is seen only in the forming operators, whose hands come into contact with the wet sizing agent.

b) Fibrous Glass and the Eye

Fibrous glass, just as any airborne dust, can get into the eye. The foreign body can be located more easily after staining with fluorescein. Although some foreign bodies may lie in the inferior conjunctival sac or on the globe, it

is not unusual to locate the offending material on the inner surface of the upper lid just posterior to the lid's margin.

c) Fibrous Glass and the Respiratory System

The effects of airborne fibrous glass on the respiratory tract have been under investigation for many years. In 1968, Wright[2] reported a study of a plant which had been in continuous production for approximately 30 years. The study included those employees who had worked in the plant for 10 or more years, some of whom had been employed for as long as 25 years. He demonstrated no unusual pattern of radiologic findings.

Pulmonary function studies by Utidjian and de Treville[3] were carried out on a random sample of 232 men from this same plant population in 1970. They included pulmonary function studies of total vital capacity, FEV, FEV_1, FEV_3 and MMF. The only correlation in these studies was age. The authors reported no apparent dust effects when comparing employees with the greatest and the least exposure to fibrous glass.

In 1971, Gross *et al*[4] studied the lungs at post mortem of 20 fibrous glass workers from the above-mentioned plant who had experienced exposures ranging from 16 to 32 years. They compared these lungs with the lungs of 26 urban dwellers who had experienced no known occupational exposures. The only significant difference found between the glass workers and the controls was a higher number of ferruginous bodies per gram of dry lung in the control group. The average total dust content, the average fibre content per gram of dry lung or per milligram of dust and the average dimensions of the fibres did not vary significantly between the control population and the fibrous glass workers.

In 1971, Nasr *et al*[5] reported on another review of chest roentgenograms from the same fibrous glass plant. No difference in the prevalence of abnormalities was detected between production and office workers.

The ability of any substance to produce or generate respiratory symptoms is related to airborne concentration. Fibrous glass, like other nuisance dusts in excessively high concentrations, may cause transitory upper respiratory irritation. Several authors[6, 7, 8] have reported upper respiratory irritation in tear-out, fabrications and manufacturing operations.

Bjure *et al*[9] performed cardiopulmonary evaluation of six insulation installers who had worked for eight to 29 years with glass wool and rock wool; they compared the results of this study with the results of a study of eight

men who had worked largely with asbestos insulating materials for seven to 30 years. The authors concluded that prolonged exposure to glass wool and rock wool did not produce impairment of the cardiopulmonary functions studied. Cardiopulmonary function abnormalities were observed in the group exposed to asbestos.

A study of mortality patterns by Bayliss *et al*[10] among 1,448 workers occupationally exposed to low concentrations of airborne fibres with a median diameter of 1.8 μ demonstrates a significant excess of non-malignant respiratory disease.

d) The Carcinogenicity of Fibrous Minerals

A number of animal studies, including intratracheal injection, inhalation and intrapleural and intraperitioneal disease of fibrous glass, have been carried out.

In 1970, Gross *et al*[11] carried out inhalation studies on rats and hamsters, exposing them to 100 milligrams/cubic meter of fibrous glass that had an average diameter of 0.5 microns and an average length of 10 microns. The exposure was carried out for six hours a day, five days a week for a period of 24 months. Uncoated fibrous glass as well as glass fibres coated with a phenol formaldehyde resin binder or with a starch binder was used. No difference in response was associated with the coatings. The results of the study were considered by the author to be consistent with the relative biologic inertness of glass. No tumours were observed.

Since 1972, Stanton and Wrench[12] and Stanton with other authors[13, 14] have been interested in the question of how a fibre causes cancer once it reaches susceptible tissues. They have approached this problem with the simple device of introducing various types of particles into the pleural space of rats and observing the resultant tumours during the subsequent two years. The methods used by them can be summarized briefly. A standard 40 mg dose of particles is applied by open thoracotomy directly to the left pleural surface of young female Osborne-Mendel rats. In each experiment, 30 to 50 rats are followed for two years, and those surviving at two years are killed. All rats are necropsied and all pathological lesions examined histologically. Tumours that resemble the mesenchymal mesotheliomas of humans generally develop after the first year. For the sake of precision, Stanton and Wrench have called these tumours pleural sarcomas. During the second year, rats died at various times with and without pleural sarcomas; consequently, actuarial computation was used to arrive

at a valid estimate of the incidence of pleural sarcomas which takes into account differences in life-span.[15] Pleural sarcomas have not been observed in several thousand untreated controls; however, they have occurred in rats treated only by simple thoracotomy. Stanton *et al*'s best estimate of these non-specific, background pleural sarcomas in treated controls is in the range of two to four per cent.

There are two separate features of asbestos particles that merit consideration as potentially carcinogenic. First, the chemical nature of their constituents and contaminants, especially those with a known potential for carcinogenicity, such as the polycyclic hydrocarbons and heavy metals. Secondly, the physical structure of asbestos particles, which in their fibrous fineness are relatively unique in nature. It is the contention of Stanton *et al* that it is the latter property, namely the simple quality of being an exceptionally fine, long, durable fibre, that is most critical to carcinogenicity.

In an experimental study by Kuschner and Wright,[16] carefully characterized glass and asbestos fibres were exposed to guinea pigs by means of intratracheal instillation. They report:

> 'Under the conditions of this experiment, there are certainly striking quantitative differences between asbestos fibre and glass fibre; but there is the strong suggestion that there are qualitative similarities. These similarities tend to support the contention that it is the physical characteristics of fibre which determine tissue damage. Indeed, one puzzling aspect is the apparent disappearance of long glass fibre from the lung. One is led to wonder whether durability of length may contribute to the quantitative differences.'

Similarly, in work done by Botham and Holt[17, 18, 19] on the development of asbestos bodies after exposure of guinea pigs to both asbestos and glass fibres, the authors report a regular sequence in the development of asbestos bodies. This sequence starts with (1) naked uncoated fibre and passes through (2) smooth coating, (3) corrugated coating, (4) partial beading of the coating, (5) complete beading and (6) fragmentation between beads.

In their series of experiments, the glass fibre bodies ran through this sequence in a significantly shorter time than did the asbestos fibres. This tends to support the speculations of Kuschner and Wright.

If the carcinogenicity of asbestos depends on its dimensional configuration, two corollary hypotheses are suggested. First, durable fibres of other materials, if in

the same dimensional range as asbestos, should be as carcinogenic as asbestos. Secondly, there should be an optimal dimensional range of fibres relevant to carcinogenicity.

Data presented by Stanton and Layard[20] at the proceedings of the workshop on asbestos during July 1977, relate to these two corollaries. The following Table lists 37 experiments with seven different durable fibrous materials, each of differing dimensional distributions, but at or near the size distribution of asbestos. Stanton and Layard[20] have listed these in order of their probability of inducing pleural sarcomas; and, as one can see, the range runs the gamut from 0 to 100 per cent. Asbestos fibres of a standard size, characterized by a working group of the *Union Internationale Contra Centre*, would fall into the range of 65 to 80 per cent.[21]

Cumulative list of experiments arranged by per cent probability of pleural sarcoma.

	Per Cent
1. Dihydroxy Sodium Aluminum Carbonate V	100
2. Potassium Octatitanate I	100
3. Potassium Octatitanate II	100
4. Silicon Carbide GTC #1	100
5. Dihydroxy Sodium Aluminum Carbonate I	95
6. Borosilicate Glass (MOL)	85
7. Borosilicate Glass (M6D)	77
8. Borosilicate Glass + Binder (KL)	74
9. Borosilicate Glass (M6L)	72
10. Aluminum Oxide - HC	70
11. Borosilicate Glass + Binder (KW)	69
12. Dihydroxy Sodium Aluminum Carbonate VII	68
13. Dihydroxy Sodium Aluminum Carbonate IV	66
14. Dihydroxy Sodium Aluminum Carbonate III	66
15. Borosilicate Glass (M6W)	64
16. Aluminum Oxide #3	44
17. Aluminum Oxide #4α	41
18. Aluminum Nitride + Oxide #6α	28
19. Aluminum Oxide #2	22
20. Borosilicate Glass + Binder (KCP)	21
21. Borosilicate Glass + Binder (KUP)	19
22. Borosilicate Glass (M8L)	14
23. Aluminum Oxide #4	13
24. Dihydroxy Sodium Aluminum Carbonate VI	13
25. Dihydroxy Sodium Aluminum Carbonate II	12

	Per Cent
26. Borosilicate Glass (MOS)	8
27. Borosilicate Glass + Binder (K2P)	8
28. Mineral Wool (Hi-Ca, Mg) (O2P)	7
29. Borosilicate Glass + Binder (KFP)	6
30. Borosilicate Glass + Binder (Y2P)	6
31. High Ca-Na (P2P)	6
32. Borosilicate Glass (M8S)	5
33. Aluminum Oxide #5	5
34. Aluminum Oxide-LC (non-fibrous)	3
35. Borosilicate Glass (YW) (vehicle) (n = 270)	2
36. Borosilicate Glass (M6S)	0
37. Nickel Titanate	0

Experimental evidences[20] suggest that particles in relatively thin (diameter $\leqslant 0.25\ \mu$) and long (length $>8\ \mu$) dimensional categories are associated with the higher tumour probabilities.

Histologic observations suggest the reason for the differences in response to fine, long fibres and those fibres that are either very short or very thick. The lesions in these experiments[20] with a low probability of pleural sarcoma were highly cellular, being composed primarily of fibroblast-laden vascular granulation tissue with a relatively low collagen content and an abundance of macrophages. In lesions from low tumour probability groups, in which virtually all fibres were less than $10\ \mu$ in length, the fibres seemed completely contained within macrophages. On the other hand, in those lesions from low tumour probability groups in which the fibres were virtually all of large diameter, the fibres seemed sequestered from adjacent tissue by both macrophages and multinucleated giant cells that closely invested the fibre surface. In contrast, the high tumour probability lesions were relatively acellular, with an abundance of collagen at the site of implantation, and the fine long fibres lay free in the interstitial tissues unaffected by phagocytes. In ancillary experiments with non-fibrous particles that stimulate collagen, such as talc, silica or carrageenin, collagen deposition equal to that of the high tumour probability lesion has been observed without the subsequent development of tumours. Therefore, it seems evident that collagen itself is not the critical factor in carcinogenesis. However, the fact that the fine, long fibres were unaffected by phagocytic activity in the high tumour probability groups suggests that fibres that are fine and long may be more carcinogenic than others, simply because they are uncompromised by phagocytic activity.

Conclusions

Both fibrous glass and asbestos are fibrous substances. As a result of the carcinogenic and fibrogenic effects of inhaled asbestos in humans, it is but natural that concern should be directed toward the evaluation of the pathogenicity of the inhaled fibrous glass.

Although experimental evidences suggest fibrogenicity and carcinogenicity of relatively thin (diameter $\leqslant 0.25\ \mu$) and long (length $> 8\ \mu$) dimensional fibrous glass in laboratory animals, it is technically very difficult to determine occupational exposure of workers to fibres that fine.[22]

Bayliss *et al*[10] have shown a significant excess of non-malignant respiratory disease among fibrous glass production workers, but in this study, no account was taken of previous employment in a dusty trade nor of exposure to silica in the batch operation of the plant under study. Pre-employment medical examinations were carried out on the population which made up the study cohort. However, during World War II, an older, and less carefully scrutinized, population was hired due to the acute labour shortage as the younger and, presumably, more vigorous, workers went into military service. Fourteen of the 19 non-malignant respiratory disease cases were hired prior to 1945.[23] A retrospective mortality study on a carefully defined group of workers in the plant under study who were exposed to finer fibre, would add important information to the health aspects of fibrous glass.

Pathological examination of the lungs by Gross *et al*[24] of long-term fibre glass workers, as well as determinations of the pulmonary mineral fibre content, disclosed that no discernible tissue reaction to fibre glass dust was present, and that the average quantity and dimensions of the mineral fibres in the lungs of fibre glass workers were very similar to those of the mineral fibres in the lungs of a control population. Authors have drawn two conclusions:

Fibre glass dust, inhaled and deposited in the lungs, causes no harmful tissue reaction.

Fibre glass dust, deposited in the lungs, is readily removed from the lung tissue by the pulmonary clearance mechanism.

The ACGIH's (American Conference of Governmental Industrial Hygienists) present Threshold Limit Value – Time Weighted Average (TLV-TWA) for fibrous glass is 10 mg/m^3, or 30 million particles per cubic foot, the so-called nuisance dust level.

All the medical research reported[25] to date has demonstrated no significant chronic health effects in

humans as a result of exposure to fibrous glass. However, in view of the demonstrated fibrogenicity and carcinogenicity of small diameter glass fibres in laboratory animals, further rapid in-depth investigations of the potential carcinogenicity of small diameter glass fibres is indicated. In the meantime, respirable exposure to small diameter glass fibres should be kept at a minimum by the use of good work practices and engineering controls.

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Chandra Bilgi, MB, BS, MD, MRCP (UK), DOH&S

Criteria for Diagnosis of Asbestosis*

Summary

The difficulties inherent in the early diagnosis of asbestosis are discussed, and the criteria upon which diagnosis is based are briefly examined.

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The first record of a case of asbestosis was reported in England by Montague Murry in 1906. The first complete description of asbestosis and of the 'curious bodies' seen in lung tissue appeared when Cooke[1] reported on a case of asbestosis, and McDonald[2] reported on the same, and another, case. Each author gave reasons for believing that these 'curious bodies' originated from asbestos fibres that had reached the lungs. Mills[3] reported the first case of asbestosis in the United States and, in the same year, Lynch and Smith[4] reported on 'asbestosis bodies' found in the sputum of asbestos workers. Early studies led many investigators to conclude that people exposed to asbestos dust developed the disease, 'asbestosis', if the dust concentration was high or their exposure was long.[5,6]

Medical science has not yet provided very much in the way of reliable and practical methods of arriving at a firm diagnosis of asbestosis in the absence of a dependable history of occupational or established exposure. As the field of opportunities for exposure widens, the possibility of asbestosis of the lungs and, particularly, changes of the pleura due to asbestos, increases. The art of diagnosis, always a matter of weighing probabilities and of looking at the total evidence, is seldom subjected to greater challenge than in the early stages of asbestosis.[7,8]

Dependence on a history of exposure for the making of a diagnosis is not uncommon in occupational medicine. It is only late in the disease that the indications add up to a virtual certainty; by then, very little can be done for the patient or for his resultant socio-economic problems.

The criteria for diagnosis of asbestosis can be considered in four categories: exposure history, functional impairment, radiographic changes and pathological confirmation.

*This paper was submitted to McMaster University as course material for the Diploma in Occupational Health and Safety.

Exposure History

A history of exposure is a most important element in the diagnostic evidence. Unfortunately, it may be quite difficult to acquire. Persons currently employed in the mining, milling, manufacture, or utilization of asbestos constitute only a part of the population at risk. Persons exposed for only short periods many years ago may not think the experience worth mentioning or they may actually have forgotten about it. Even short periods a couple of decades ago, when controls were not so stringent, may have been quite sufficient for pathogenesis to begin. Such short exposures are easily overlooked, but they cannot be lightly dismissed. It must also be remembered that the possibility of neighbourhood or family exposure exists, in persons working around, but not in, the actual handling of asbestos, as in shipyards or building operations.

One cannot neglect the fact, either, that there are over 3,000 asbestos consumer products on the market that may permit exposure in the course of their manufacture, use or disposal.

Functional Impairment

Even in fairly early stages of the disease, there are some indicators that can be sought once a suspicion of exposure has been aroused or in the course of a routine examination of exposed personnel. One that sometimes develops quite early is a reduction in the carbon monoxide diffusion capacity of the alveoli,[9, 10, 11] which may be detectable before any complaint of dyspnea is offered. Changes in compliance of the lungs are considered by some to provide an early suggestive sign.[12] A reduced rate of lower lobe emptying in expiration, as revealed by an admixture of radioactive xenon, is advocated by Stanley.[13] At a somewhat later stage, dyspnea accompanied by reduced vital capacity and lung volume, and the presence of persistent, fine rales at the bases of the lungs, constitute a very suggestive combination, even in the absence of demonstrable radiographic changes.

Later in the disease, clubbing of the fingers is often seen and cyanosis may be present. By this time, however, dyspnea is often marked, and radiographic appearances consistent with the diagnosis have usually appeared.

Radiographic Changes

For systematic evaluation of chest radiographs in suspected cases of asbestosis, the UICC classification, developed for silicosis and coal workers' pneumoconiosis, has been modified to the form known as the ILO–U/C classification, and is particularly useful for the recording of pleural changes and irregular parenchymal infiltrates.[14]

Parenchymal changes provide few radiographic features that are characteristic of asbestosis, but some are highly suggestive, particularly when they occur together. The irregular, reticular, and nodular changes are more commonly seen in the lower two-thirds of the lung fields, and are generally more easily visible on the right side. The apices remain lucent, and massive confluent shadows are uncommon. Hilar enlargement is not usually marked until pulmonary hypertension develops. A shaggy heart outline is not rare; interlobar septa may be visible, and shadows, described as resembling Kerley 'B' lines, may appear. As lung volume diminishes, the apicocaudal diameter is diminished, the costophrenic angle is narrowed and the lesser interlobar fissure descends.

Pleural thickening may lead to obliteration of the costophrenic and, sometimes, of the cardiophrenic, angles, and to tenting of the diaphragm, although the latter may be found without adhesions. The pleural fibrosis may appear as soft tissue shadow between the lung periphery and the chest wall, and adhesions may distort the lung shadow. This is particularly true in the region of the lingula or mid-lung fields.

Hyaline pleural plaques may appear as faint diffuse shadows that persist in repeated films when viewed *en face*, and may be seen a little more clearly when viewed end on.

The presence of calcified pleural plaques, distinguished from intrapulmonary calcification, is practically pathognomonic of exposure to asbestos, talc or, occasionally, mica, especially in the absence of past thoracic hemorrhage, trauma or tuberculosis. Calcified plaques commonly appear bilaterally and in generally symmetrical arrangement as irregular patches, mainly in the middle and lower zones. However, unilateral plaques are not uncommon.

Asbestos exposure must be considered when pleural effusion, free or loculated, has come on quietly, particularly if it undergoes spontaneous resorption. The question of mesothelioma also enters into the differential diagnosis of this condition.

Pathological Confirmation

While the sum of clinical evidence may strongly suggest the disease and warrant the diagnosis in an occupationally exposed person, in most instances it will be rendered unequivocal after microscopic examination of lung tissue obtained by trans-bronchial biopsy, needle biopsy, open chest biopsy or autopsy. The extent to which surgical intervention is justified in the search for an unequivocal diagnosis is a matter for clinical judgement in each individual case. In general, intervention is not essential. The appearance of typical interstitial alveolar fibrosis accompanied by numerous asbestos bodies will serve to confirm a diagnosis that functional and radiographic evidence may have left equivocal, but the presence of asbestos bodies or even asbestos fibres alone only confirms exposure, but is insufficient evidence for reaching the diagnosis of 'asbestosis'.

The histological appearance of pleural thickening, while highly suggestive, is not specific for asbestosis, but the presence of numerous asbestos bodies, or fibres, in the tissue or in contiguous parenchyma will serve to support the diagnosis. The presence of hyaline, and, particularly, of calcified, plaques in the typical locations mentioned above, would very strongly support the diagnosis.

Conclusion

Asbestosis is sooner over-diagnosed than missed. Incorrect diagnosis may result in unfounded socio-economic and psychological damages and/or the danger that treatable diseases (e.g. rheumatoid lung) may remain untreated.

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Malignant Mesothelioma: An Asbestos-Associated Tumor

Summary

This paper presents a brief review of malignant mesothelioma and its relationship to asbestos exposure. The risk of developing this serious disease is related to the severity of the exposure, but it is not yet possible to quantitatively estimate the risk at any given exposure level.

Malignant mesothelioma is a relatively rare tumour which has gained notoriety in the past 20 years because it has been observed to be associated with occupational and environmental exposures to asbestos dust.[1] In this article, I will present a brief discussion of this tumour, its occurrence in Canada and its relationship to asbestos exposure.

The Pathology of Malignant Mesothelioma

Malignant mesothelioma is a tumour that arises from the tissues lining the chest (pleural) or abdominal (peritoneal) cavities. It grows to ensheath and constrict the internal organs, interfering with their function. Microscopically, malignant mesothelioma consists of a mixture of two types of tissues, one of which might predominate in any individual case. Mesothelial cells have the potential to develop as either mesenchymal stromal cells or epithelium-like lining cells. The mesenchymal type of mesothelioma appears as a spindle-cell sarcoma, while the papillary type consists of cuboidal, columnar, or flattened cells forming tubular and papillary structures resembling adenocarcinoma. Poorly differentiated mesotheliomas may be exceedingly difficult to differentiate from carcinoma.[2].

The development of mesothelioma is devastating. There is, at the moment, no effective therapy and no cure, and death usually occurs within a year or two of diagnosis.

Malignant Mesothelioma in Canada

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It is impossible to accurately determine the number of cases of mesothelioma occurring in Canada. The tumour is rare and difficult to diagnose and probably is occasionally not recognized as being mesothelioma. Furthermore, the diagnosis of mesothelioma on the death certificate is occasionally coded as some other condition and will thus not be appropriately classified in provincial vital statistics. In a study of mortality among workers receiving Workmen's Compensation for asbestosis in Ontario,[3] we found that none of the three pleural mesotheliomas recorded on death certificates were coded as pleural tumours, and they would thus not appear as

A group at McGill University attempted a complete ascertainment of all fatal cases known to Canadian pathologists in the years 1960-75.[4] They wrote to all members of the main pathologic societies in Canada enquiring about all fatal cases of primary malignant mesothelial tumours diagnosed by means of autopsy or biopsy and continued their enquiries until a reply was received from every pathologist. They found that during the first half of the 1970s there were about 20-30 fatal cases per year across Canada. They used a similar approach to ascertain the number of fatal cases occurring in the United States during 1972 and, combining the Canadian and American statistics, they found that 70 per cent of the cases occurred in males, and that pleural tumours were two to three times more frequent than peritoneal tumours.

At the time of death, 11 per cent of the males were under 45 years of age, 50 per cent were 45-64 years old and 39 per cent were 65 years of age or older.

In Ontario, the annual incidence rate during the years 1966-75 was 5.7 deaths per million men aged 45 years and over, and 2.2 deaths per million women aged 45 years and over.

Among workers, the Ontario Workmen's Compensation Board has, to date, accepted about 30 claims for mesothelioma.

The Association with Exposure to Asbestos

In 1960, J.C. Wagner[1] reported 33 cases of this tumour in South Africa, of which 13 cases occurred in crocidolite asbestos miners and millers and 14 in persons living in the neighbourhood of these mines. Since then it has been found that a sizeable fraction of people developing mesothelioma have had exposure to asbestos and that populations of workers exposed to asbestos dust have had much higher than expected mesothelioma mortality rates. Experimenters[1] have been able to induce mesotheliomas in animals by exposing them to air-borne or injected asbestos dusts. Not all cases of mesothelioma can be related to asbestos exposure, however, and there must be spontaneous cases or cases related to some other causative agent.

It is suspected that not all types of asbestos may be equally potent in producing mesotheliomas. This suspicion is based primarily upon the observation of only 10 mesotheliomas among the miners and millers of chrysotile asbestos in Quebec.[1] It is difficult, however, to compare the health patterns of workers who have experienced different exposure conditions, and this differential toxicity has not been firmly established.

There is frequently a long interval of time between first exposure to asbestos and death from mesothelioma. This interval is usually greater than 20 years and has occasionally been as long as 50 years or more. This observation is important from the industrial hygiene viewpoint since cases of mesothelioma now occurring are related to the exposure conditions of many years ago; the effect, if any, of current workplace conditions upon the health of workers would thus not be expected to be detectable for many years to come.

Workers at the highest risk of mesothelioma have been insulators, factory workers who have worked in the production of asbestos products, workers in the heating trades, and shipyard workers. The degree of risk is probably related to the magnitude of the exposure, and this will be discussed in the next section.

The Dose-Response Relationship for Mesothelioma

A dose-response relationship is a description of the relationship between the dose of a particular agent and the risk or probability of a particular outcome (sickness or death). Because some people who have developed mesothelioma have been estimated to have had only low exposures to asbestos dust, the claim has occasionally been made that there is no dose-response relationship for mesothelioma. I believe that this statement is false and that there is reasonable evidence to lead one to believe that the risk of developing mesothelioma is related to the magnitude of the exposure to asbestos. If one accepts the hypothesis that most of the cases of mesothelioma that occur are related to exposure to asbestos, then the cases occurring after what would seem to be trivial exposures would suggest that there may be no lower threshold of exposure for induction of this malignancy.

Whitwell[5] and his colleagues in England measured the concentration of asbestos fibres, visible by polarized light optical microscopy, in the lungs of 100 mesothelioma victims, 100 lung cancer patients and 100 other 'controls'. The three series are not strictly equivalent in terms of age and sex distribution, but the authors did compare them and found that the mesothelioma victims generally had much higher concentrations of asbestos in their lungs. The implication is that individuals who have experienced ambient or low exposures are at much lower risk than those with larger exposures. They also found that some of the women with mesothelioma who had experienced brief, but presumably intense, exposures many years previously, still retained large quantities of fibres in their lungs.

Newhouse and Berry[6] have found that mesothelioma mortality rates among workers at an asbestos factory in England increased with increased estimates of the severity of exposure, and a similar pattern has been observed among long-term employees of an asbestos factory in Ontario.[7] It is unfortunately not yet possible to define the shape of the dose-response curve or to give a quantitative estimate of the risk at any exposure level.

Conclusions

Malignant mesothelioma is a rare but lethal tumour associated with exposure to asbestos; it is probable, however, that there are spontaneous cases or cases related to some other causative agent. The magnitude of the risk of developing this tumour is related to the severity of the asbestos exposure, but the shape of the dose-response curve has not yet been defined and it is not possible to quantitatively estimate the risk at any given exposure level. There is ongoing research around the world, and one can anticipate increased knowledge about this disease and its causation.

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Your New Occupational Health Laboratory

The Occupational Health Laboratory of the Ontario Ministry of Labour moved into its new premises on April 1st, 1981. The high standards and scientific proficiency inherent in the new Occupational Health Laboratory are the culmination of 60 years of successful work in the field of industrial hygiene.

The first laboratory was established in 1921 in the Whitney Block at Queen's Park by the Division of Industrial Hygiene under the Ontario Board of Health. After operating within a variety of departments, it was moved, in 1951, to temporary quarters in an old building on Christie Street, but remained in its temporary home for a total of 30 years. It has been known as the Occupational Health Laboratory since 1969.

Industrial health hazards were only dimly perceived in the 1920s, but the laboratory helped to increase awareness and developed improved methods of evaluation. The list of identified hazards grew enormously over the years, and techniques for sampling and analysis gradually became extremely sophisticated.

Increases in the volume of work carried out by the laboratory over the last few years provide ample evidence of growing concern with occupational health. For example, during the twelve-month period ending March 31st, 1981, the number of analyses performed exceeded 64,000 – in contrast to under 34,500 in the 1977-78 period.

The laboratory provides full-scale analytical support for the Occupational Health Branch Medical and Hygiene Services and may, at the discretion of the director, provide specialized service to other agencies. Its comprehensive range of current instruments and equipment provides capabilities of analysing a wide range of inorganic substances, organic compounds and particulate matter. The analytical instruments include atomic absorption spectrometers, a plasma emission spectrometer, electron microscope, mass spectrometer, fourier-transform infra-red spectrometer, spectrometer fluorimeter, gas and liquid chromatographs plus polarography and X-ray diffraction equipment. Many of the instruments are automated and computerized to speed up completion of analysis and to eliminate elementary,

The new Occupational Health Laboratory of the Ontario Ministry of Labour.

Preparation of samples for analysis is carried out in rooms designed for that purpose. These wet chemistry laboratories are isolated from equipment used for final analysis.

but time consuming, labour, thus releasing highly skilled staff for development of improved methods.

The laboratory performs analyses of samples varying in size from bulk to trace quantities. The analysis of samples submitted as part of the routine medical surveillance of workers exposed to potentially hazardous substances continues to be a major function of the laboratory. It is also committed to the development of new, or refined, analytical methods to cope with the increasing number of complex chemicals and mixtures of chemicals that will be used throughout industry in the years to come.

From energy efficiency to stringent pollution control, the new laboratory building was designed for the specialized nature of its work. Safety precautions and safe working procedures are in force everywhere. A modern security system monitors all areas, and gases used in the laboratory are piped from a central, but external, storage bay. Sample receiving facilities provide correct handling and storage of all samples. The Occupational Health Laboratory, the largest and most extensively equipped facility of its kind in Canada, is one of Ontario's most important occupational health resources, and will undoubtedly play an important role in protecting the quality of life in the workplace.

Respirable fibres of asbestos collected from air passed through a filter are counted by optical microscopy. In this way fibres of toxicological interest are measured directly.

Inductively Coupled Plasma Emission Spectrometer measures the light emitted from metal atoms recovered from blood, urine or air samples for up to 31 different metals all at the same time.

Liquid chromatography is used to detect chemicals which may decompose during analysis by other methods. Injected mixtures are separated into individual compounds and analysed by appropriate detectors.

Book Review

by Murray Grant
3rd edition — 1981
Publishers: Lea and Febiger, Philadelphia, PA
Price $14.50 in Canada
368 pp

Handbook of Community Health

This excellent paperback reviews basic principles and practices of public health and preventive medicine. The principles are general, but description of the practices and legislation focuses on the United States of America. Although new chapters have been added and old ones updated, this third edition retains an antique flavour, not mentioning such important recent events as the eradication of smallpox and the decline in coronary heart disease mortality in the USA. But when dealing with a field as large as community health in an introductory text for non-specialists, it is difficult to decide what to leave out and what to include. Overall, this book achieves the balance. Students of pharmacy, dental, nursing and paramedical programs will find this book most valuable because it covers the subject so clearly and concisely. It is recommended.

M.J. James

Publications Available from Government Book Store

Publications under the Regulation Act vol. 112-39	
O. Reg. 658/79 as amended by O. Reg. 844/79 Industrial Establishments	$1.50
O. Regs. 659/79 as amended by O. Reg. 845/79 Construction Projects	$1.50
O. Regs. 660/79 as amended by O. Reg. 846/79 Mines and Mining Plants	$1.50
The Nursing Consultant	$2.00
A Guide to The Occupational Health and Safety Act, 1978	$1.00
Regulation respecting Lead — made under the Occupational Health and Safety Act Revised Statutes of Ontario, 1980 Chapter 321	$1.00

News and Notes

Occupational Health Seminar, Toronto June 22-23, 1981

As part of the educational effort of the Ontario Section, AIHA and IAPA co-sponsored a two-day program of lectures and workshops designed to provide a practical working guide to occupational health in the work environment. About 179 participants attended the seminar. Among the attenders were plant managers, production managers, engineers, safety and health personnel and labour representatives.

Papers were presented by the leading authorities in industry, government and educational institutions. Concurrent workshops were held on Air Sampling Techniques, Asbestos Hazards and Control, Training and Communications in the Plant, Noise Measurement and Control, Respiratory Protection, and Welding Hazards. The preliminary sessions included papers on Health Hazards in the Workplace, Basic Principles of Industrial Hygiene, Occupational Health and the Law, Hazard Identification and Evaluation, Principles of Engineering Controls, Health Surveillance, Biological Monitoring, and An Effective Occupational Health Program.

Occupational Medicine Section of the Academy of Medicine, Toronto

The meeting dates will be on the first Thursday of the month as follows:

1981 October 1st
December 3rd

1982 January 7th
February 4th
April 1st
June 3rd – Dinner

The topics will be announced in the Academy Newsletter.

The committee for the session 1981/82 is as follows:

Chairman	— Dr. M. Kerr
Secretary	— Dr. PL. Pelmear
Program Co-ord.	— Dr. G. Debow
Members	— Dr. J. Nethercott
	— Dr. E. Kaegi

The annual report of the Advisory Council on Occupational Health and Occupational Safety, detailing 38 recommendations in four key subject areas, was released on June 16, 1981.

The recommendations are contained in four advisory memoranda, either initiated by the council or in response to requests from the minister. The minister's responses are included.

The four topics dealt with specifically in the report are:

- policies and principles for manpower planning and educational programs in the field of occupational health and occupational safety
- policies and principles concerning research and development in the field of occupational health and occupational safety
- procedures for monitoring and maintaining occupational health and occupational safety standards in the workplace: accreditation, external and internal audit programs
- policies and principles for the use of information in the prevention of occupational accidents and illnesses.

Reporting its findings on the topic of manpower planning and educational programs, council recommended that educational programs oriented toward those currently employed should be given funding priority. It underlined the need to ensure that occupational manpower data are available about different health and safety skills needed in Ontario, and recommended that education and training programs should, wherever possible, be delivered through joint co-operative arrangements among post-secondary institutions utilizing local industrial expertise.

In its report on the use of information to prevent occupational accidents and illness, the council pointed to the need for a system of uniform data-collection within the province. It suggested that protocols be developed for the detailed causal investigation of those accident types that are responsible for the greatest degree of human injury and disability.

The council's Third Annual Report also outlines its discussions in Thunder Bay with representatives of management and labour from the forest products and pulp and paper industries in that region.

Pulmonary Function Testing

A course entitled Pulmonary Function Testing in Industry was given on June 22, 23 and 24, 1981 as a new venture by the School of Continuing Studies, University of Toronto, in co-operation with the Canadian Society of Pulmonary and Cardiovascular Technologists and the Occupational and Environmental Health Unit of Preventive Medicine, University of Toronto.

Many companies are becoming interested in monitoring the respiratory health of their employees at the work site, and are trusting this responsibility to persons who have had little opportunity for training in the field. In recent years, there have been strong movements to standardize the performance of these tests. While such requirements have become legally mandatory in the United States, no firm certification requirements as yet exist in Canada. The current course is the first in an attempt to improve the general level of lung function testing in the province. During a three-day intensive study program, participants, consisting almost entirely of occupational health nurses, learned the basics of lung anatomy and physiology, and the patterns of both general and occupational respiratory diseases as they appear reflected in the spirometric tracing. Information was given on how to handle a variety of instruments, how to cope with the individual who takes bronchodilators, and how to calculate the results and convert them to standard reporting modes. Calibration techniques and a discussion on infection control and reference values completed the theoretical instruction. Interspersed between lectures were four workshops in practical training.

The overall response was very gratifying, and it has been decided to repeat this course. The next one is expected near the end of November 1981. The course organizers for the first venture were Dr. J. Roos of the Occupational and Environmental Health Unit, University of Toronto and Mrs. J. Bernard of the Canadian Society of Pulmonary Cardiovascular Technologists.

Coming Events

October 4-9, 1981	2nd World Congress of Chemical Engineering and IX Interamerican Congress of Chemical Engineering, Montreal, Quebec.
October 19-23, 1981	A Short Course in Occupational Health, the first in a series of courses particularly designed to meet the needs of part-time physicians in industry, is being offered by the University of Toronto Faculty of Medicine. Registration fee, $250. For applications and further advice write to: The Director, Occupational and Environmental Health Unit, Faculty of Medicine, University of Toronto, 150 College Street, Toronto, Ontario, M5S 1A1. Tel. (416) 978-4353.
October 19-23, 1981	Ontario Occupational Health Nurses Association, 10th Annual Conference, London, Ontario.
October 25-29, 1981	47th Annual Scientific Assembly, American College of Chest Physicians, San Francisco. Further information may be obtained by writing to the American College of Chest Physicians, 911 Bussye Highway, Park Ridge, Illinois, 60068.
October 29-30, 1981	Society of Occupational Medicine, Autumn Meeting and AGM., London, England.
November 11-14, 1981	Ontario/Quebec Conjoint Meeting OMA Section on Occupational Health, Montreal, Quebec.
November 23-25, 1981	2nd International Congress on Analytical Techniques in Environmental Chemistry, Barcelona, Spain.
March 7-11, 1982	Annual Meeting of the Society of Toxicology, Boston, Massachusetts.
April 25-30, 1982	American Occupational Health Conference, Toronto, Ontario.

Indices

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